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ARTERIO-SCLEROSIS AND THE EYE

THE RICHARD MIDDLEMORE POSTGRADUATE LECTURE, 1919*

BY

P. H. ADAMS, M.A., M.B., D.O.OXON., F.R.C.S.,

OXFORD.

I MUST first express my gratitude to those who have done me the honour to invite me to deliver the Middlemore Postgraduate Lecture this year. The idea of the lecture, I believe, is that the subject chosen should be one that is of interest to all members of the profession and it is with that end in view that I have selected as my subject arterio-sclerosis and the eye.

There can be no question, I think, as to the extreme importance of this subject to our patients, to the practitioner of general medicine, and to the ophthalmic surgeon.

In considering the subject of arterio-sclerosis the first point of importance to remember is that this is not a disease of itself, but rather the result of several different pathological processes, some of which are but dimly understood.

This subject is best divided into three groups:

First groub, called Hyperpiesia by Clifford Allbutt,[1] characterized by simple high tension, without signs of arterial or renal disease.

* Delivered on December 2, 1919, at the Birmingham and Midland Eye Hospital.

Second group: Arterio-sclerosis with associated high tension, renal, and heart changes.

Third group: Chronic nephritis, with secondary high pressure, arterio-sclerosis, and heart changes.

Hyperpiesia.—In many ways this is the most important group of cases from our point of view, for reasons which I shall endeavour to show.

It was Clifford Allbutt[1] who recognized that high pressure preceded actual arterial changes, and it is to this condition that he gives the name of hyperpiesia.

If treated appropriately, he states, it may be cured, or at any rate recur with less persistence. On the other hand, if neglected, it tends to recur and to progress, and ultimately to become associated with true arterio-sclerotic changes. These facts show that high arterial blood pressure is at any rate one of the earliest signs of arterio-sclerosis; and if not a direct cause, is, seeing that it may be made to disappear without arterio-sclerosis resulting, at any rate associated with conditions which give rise to this condition.

Williamson[2] in a very practical paper on this subject, states that it is now generally recognized that in hyperpiesia, the increased resistance to the blood flow is associated with a contraction of the muscular coat of the medium and smaller arteries, causing a diminution of their lumen. This fact was first emphasized by W. Russell[3] and called by him "arterial hypertonic contraction."

Russell's explanation of the phenomenon is as follows:

Toxic substances cause a hypertonic contraction of the arterioles and of the arteries, from as large as the brachial downwards. That an increased pressure is thus caused, which if recurring or continuous, leads to hypertrophy of the muscular coat of the arteries.

"If hypertonus persists, it must lead to hypertrophy, hypertrophy being the normal result of such a condition."

According to Osler[4], the changes in these cases are not in the heart, or main drainage system, nor in the supply pipes, but to keep up the normal irrigation in the capillary beds, there has to be a widespread increase in pressure in the smaller arteries. He believed that the primary mischief lay between the capillary wall and the lymph space, or as he graphically put it: "There is a row between the manufacturer, the middle man, and the sanitary authorities." In consequence of this, an increase of pressure is necessitated in the supply pipes to overcome this.

Mackenzie[5] states that the arterial pressure is maintained by the force of the left ventricle, the peripheral resistance, and the elastic recoil of the arteries, and unless the pressure is kept up there is not a regular and sufficient supply of blood to the tissues. He

believes that there is an intimate association between the heart and the tissues, whereby the supply is moderated to their requirements.

As age advances three chief changes occur in the blood-vessels—

(1) The elasticity of the arteries diminishes, *i.e.*, they become more like rigid tubes and there is no longer the same equable pressure during diastole. Therefore the pressure must be increased during the ventricular systole to compensate for this.

(2) There may be increase of the muscular tissue in the arterioles, which implies an increase in functional activity, with increase in the peripheral resistance.

(3) There is great diminution in the capillary fields, owing to thinning and wasting of the skin and subcutaneous tissue, causing a considerable narrowing of the passage of outflow.

According to Mackenzie[3], the conditions which induce degenerative changes in the arterial system are—

(1) High blood pressure;
(2) Toxic conditions;
(3) Senile changes;

and he lays special stress on the obliteration of the capillaries, which causes degenerative changes in the heart, and narrows the communication between the arterial and venous system, entailing more work on the heart in forcing blood through the constricted area.

In what type of patient, then, is it that these changes are most liable to occur?

Osler[4] says it is the man who works hard, drinks hard, and smokes hard, particularly the keen business man with many responsibilities, and also occasionally gouty or neurasthenic persons. To these, one may truthfully add, I think, the man who plays hard and loves hard. In fact it is the penalty of the man who adopts as his life's motto: "Whatsoever thy hand findeth to do, do it with thy might."

The next point to consider is the symptoms that such patients complain of.

Williamson[2] gives the following, which I have arranged in two groups:

(1) Heaviness or fulness of the head, drowsiness, insomnia, and fatigue especially in the morning. Inability to concentrate, despondency, giddiness, noises in the ears, migraine, neuralgia, and nervousness.

(2) Local syncope of the extremities, rheumatic pains or cramps in the limbs, tendency to haemorrhage, haemoptysis or epistaxis, dyspnoea on exertion, and intermittent polyuria.

On thinking over this list, we find that we, as ophthalmic surgeons, are frequently consulted for many of them, on the supposition that they may be caused by eye strain or want of glasses.

It is the symptoms placed in the first group, which are more usually complained of by our patients, and I should like to add to them, headaches on waking in the morning, and pains in the muscles at the back of the neck. The headache that patients complain of when they first wake, or that even wakes them up in the night, but passes off after a cup of tea and getting up, is, I believe, never caused by the eyes and always raises a suspicion of high blood pressure in my mind. At the same time it is not an unusual complaint of women at the menopause. Pain at the back of the neck again may be caused by errors of muscle balance, particularly exophoria, but this is more so in young subjects, and when occurring for the first time in middle age, is, I believe, a symptom suggestive of raised pressure.

Cases of migraine again are frequently sent to the ophthalmic surgeon to discover if the attacks are caused by eye strain. In my experience, it is the exception, rather than the rule, to find an error of refraction sufficient to account for the condition. I am not implying that migraine is usually caused by arterio-sclerosis, but in those patients who have their first attack somewhere about the age of 50, with tendency to frequent repetition, I believe the trouble often lies in heightened blood pressure.

Giddiness is another symptom which, to my mind, is rarely caused by eye strain or refractive error, though sometimes patients with astigmatism will complain of this, and are relieved by appropriate glasses. Those cases of giddiness which come on, on change of position, such as getting up after sitting in an easy chair for some time, or getting out of bed in the morning, seem obviously due to circulatory disturbance, and yet I have had many people sent to me to have their eyes tested for glasses on account of this symptom, the confusion arising, I believe, from the fact that the patient was reading in the chair before getting up.

Williamson[2] also draws attention to the fact that the symptoms of hyperpiesa may be mistaken for neurasthenia.

First, then, we see that there are many symptoms of hyperpiesia which are liable to be confused with those caused by eye strain.

Secondly, all the medical authorities whom I have consulted agree that the most important consideration, as regards the prognosis and treatment of the hyperpietic condition, is its early recognition.

Thirdly, we, as ophthalmic surgeons, see an enormous number of patients between the ages of 40 and 55, the common age of its occurrence, for the purpose of ordering presbyopic glasses.

Thus, a heavy responsibility is ours if in the ordinary routine examination of presbyopic subjects, we miss a case of hyperpiesia, and are content to order a pair of reading spectacles, without going

further into the case when the symptoms are suspicious, and the eyes insufficient to account for them. Is it possible, then, to diagnose from the condition of the retinal arteries whether hyperpiesia is present?

Bardsley[6] states that in simple high tension the following manifestations may be found:

(1) The vessels have an appearance of uniform distension and fulness.

(2) The light streak is broadened out, it may be greatly increased, apparently reaching almost the whole breadth of the vessel.

(3) The light streak is very much brighter than normal, the brilliancy increasing with the increase of tension, until with very high tension it becomes like bright copper wire (not silver wire).

(4) The tight arteries indent the veins; with medium high tension they indent them slightly, with very high tension they indent them deeply, leading to back pressure and its consequences.

The following signs, he states, indicate actual sclerosis:

(1) Irregular tortuosity, especially of the smaller twigs.

(2) Increased brilliancy of the light streak, while at the same time the light streak appears narrower and more central.

(3) Irregularity of the calibre and beading of the arteries.

(4) General diminution in the size of the vessels and silver wire reflex show advanced sclerosis.

He confirmed his results by the injection of solutions of adrenalin chloride on healthy adults, the difference in the vessels before and after injection being noted.

He goes so far as to say that it is possible to indicate roughly the actual height of the blood pressure in mm. of Hg. from the observation of the retinal vessels alone.

Where there are no signs of sclerosis, he says:

(*a*) Full arteries with broad light streak and very slight indentation of the veins indicate a blood pressure of 140-150 mm. of Hg.

(*b*) Very brilliant broad light streak with deeper indentation, and slight signs of obstruction indicates 150-160 mm. of Hg.

(*c*) Very brilliant broad light streak with apparent obliteration of the veins, indicates a pressure of 160-180 mm. of Hg.

But where the signs of advanced sclerosis are present, the veins may be indented and nipped at lower tensions.

Bardsley also emphasizes the pitfalls that may arise; namely (1) High error of refraction. (2) Acute toxaemia. (3) Failing heart.

Keeping these three liabilities to mistake in mind, Bardsley thinks it is possible for the ophthalmic surgeon to distinguish by means of the ophthalmoscope the signs of increased blood pressure and to differentiate between that and arterio-sclerosis.

Personally, whilst recognizing the value of Bardsley's observations

I have not found them so universally true as one had hoped; perhaps it is not fair to expect this.

First, you may get a very much higher blood pressure than he mentions, without any gross retinal signs. To illustrate this I may quote the following case:

Mrs. C. B., aged 52 years. She woke with headache in the night and every morning rose with one. She was sent to me by her doctor for glasses. She had, however, only a slight error of refraction. On examination of the fundi, the arteries showed no sign of disease, and the only suggestion of raised blood pressure that I could find was that the veins lost their light reflex for a short distance above where the arteries crossed them. There was no indentation or pinching of the veins, and no exaggeration of the light reflex, and yet I found her blood pressure to be from 195 to 200 mm. of Hg. and treatment directed to the control of this was the means of relieving her headaches. When I saw her some six months later, she said she felt a different woman, that I was the only person who had been able to find out what was the matter with her and she was correspondingly grateful. As a matter of fact I diagnosed the condition chiefly from her symptoms and the manometer and not from her retinal arteries.

A converse case to this was:

A lady, aged 53 years, complaining of constant headaches, the retinal arteries appeared definitely thickened, and yet her blood pressure was only 130 and she had not got a failing heart or acute toxaemia.

Again, some children, especially those with a good deal of hypermetropia, have full vessels with quite a pronounced bright reflex sometimes associated with considerable tortuosity of the arteries without any raising of the pressure or sign of arterio-sclerosis.

Thus, whilst not decrying Bardsley's observations, I do not think one can, and, of course, no one in practice would, rely on the ophthalmoscopic signs only, for a reliable estimate of the blood pressure.

To practise one's skill in the estimation of blood pressure and the presence or absence of any abnormality, I examined some cases, selected at random, with the ophthalmoscope and afterwards had their blood pressures taken by a second person.

Eighteen cases, varying in age from 48 to 67, I passed as normal, or within normal limits, as regards their retinal vessels.

Of these:

9 had blood pressure of 140 mm. of Hg. or under
2 „ „ „ „ 150 mm. of Hg. aet. 52-53
1 „ „ „ „ 160 mm. of Hg. aet. 53
4 „ „ „ „ 170 mm. of Hg. aet. 60, 64, 50, 56
1 „ „ „ „ 180 mm. of Hg. aet. 67
1 „ „ „ „ 170 mm. of Hg. aet. 49, but I marked her as a little doubtful.

According to Oliver[7], the average normal pressures

between 40 and 60 = 125 to 135 mm. of Hg.
between 60 and 80 = 135 to 165 mm. of Hg.

adding or subtracting 15 mm. to get the outside limits of normal variation.

So the two cases of 59 and 56 with a blood pressure of 170 and the doubtful case of the woman of 49 with a blood pressure of 170, are the only cases that were badly outside possible normal limits, yet whom one passed as normal as regards their retinal circulation.

Probably every man practised in the examination of retinal blood vessels looks out for some particular sign more than another, and comes to lay more stress on this than any other, and I must admit that the sign of heightened pressure which has impressed me most, is the small size and pale colour of the arteries, quite apart from thickening or exaggeration of the light reflex, and I believe you can safely say that you never see a nice fat artery with an ordinary light reflex or even an exaggerated light reflex in high blood pressure. This is rather what one would expect, remembering Russell's observation on arterial hypertonic contraction.

Again, where an artery crosses a vein in a normal patient, the light reflex of the latter is continuous practically up to the artery, but if the pressure is raised, before one sees obvious pinching or indentation at the crossing, there is an absence of light reflex for some short distance up and down the vein, due I suppose to some flattening of the curvature with consequent alteration of the reflex.

It is these two symptoms, then, which to me show the earliest indications of an elevation of the blood pressure pure and simple, before the onset of any signs of actual sclerosis.

Williamson[2] in his lecture says that it is necessary to emphasize the importance of persons of middle age having their blood pressure measured at intervals in order that the early development of hyperpiesia may be detected and that arterio-sclerosis may be prevented. I rather think that the ophthalmoscope is in many ways a more accurate instrument than the manometer, and certainly will give more information as to the exact condition of the smaller blood-vessels, considering the fact that the retinal arteries can be examined in their natural condition during life under a magnification of 15 diameters by direct ophthalmoscopy.

Group II

Arterio-sclerosis with associated high tension, renal and heart changes.

Hitherto we have been considering the pre-sclerotic stage of arterio-sclerosis, *i.e.*, before the disease has persisted sufficiently long or progressed so far as to cause permanent damage to the

blood-vessels. We must now consider the more advanced changes that we meet with, when actual and permanent damage has occurred.

Here it is necessary to refer to the classical work of Marcus Gunn.[8] He was the first in this country to draw attention to the importance of and to describe accurately the changes in the blood-vessels met with in this condition.

His work is so well known to you and has been quoted so often that it is not necessary here to do anything more than enumerate the various signs that he described. These observations have been universally confirmed, and their extreme value recognized again and again.

They were as follows:

(1) Irregularity of the lumen of the retinal vessels.

(2) Tortuosity of the arteries.

(3) A very narrow and bright central light streak which may show a series of brighter points at intervals. These changes being of chief importance, when they involve the secondary and tertiary branches of the retinal artery.

(4) Loss of translucency of the arterial walls.

(5) Obstruction of the blood flow in the veins where they are crossed by arteries.

(6) Oedema of the retina.

The importance of some of these signs has proved to be greater than others and a few more have been added, but they still remain the fundamental and classical signs of arterio-sclerosis.

de Schweinitz,[9] another authority on this subject, divides the signs of persistent high tension into those that are suggestive, and those which are pathognomonic.

The suggestive signs are: A. Uneven calibre and undue tortuosity of the retinal arteries; B. Increased distinctness of the central light streak; C. An unusually light colour of the breadth of the artery and alterations in the course and calibre of the veins.

The pathognomonic signs: A. Changes in size and breadth of the retinal arteries of such character that a beaded appearance is produced; B. Distinct loss of translucency; C. Decided lesions of the arterial walls and white stripes of perivasculitis; D. Alternate contractions and dilatations of the veins: indentations of the veins by stiffened arteries in such a way as a solid rod would indent a rubber tube when lying across it. The vein may be:

(*a*) flattened slightly, (*b*) pushed aside, (*c*) its calibre contracted, so that beyond the point of crossing there is an ampulliform dilatation.

In addition there are changes in the venous walls, white stripes, oedema of retina, linear extravasations, and haemorrhages.

de Schweinitz[9] also lays stress on the fact that these changes may occur at any age, provided the raised blood pressure is persistent.

According to him, the earliest signs are :

(*a*) Corkscrew appearance of certain twigs, either round the macula or small branches of larger vessels, especially single vertical twigs from a transverse branch.

(*b*) When a vein crosses over an artery, it is like a strap over a solid tube, and when the vein passes under an artery it is like a solid tube over a strap. The changes are first noticed in the inferior temporal artery, but personally I have found them as often in the superior temporal artery.

(*c*) A congested appearance of the optic disc, giving it a dull red appearance,

Foster Moore[10] lays most stress on Gunn's fifth sign, i.e., obstruction of the blood flow in the veins where they are crossed by arteries, but with emphasis more on the course of the vein rather than on any obvious sign of damming up of the blood, which is found very rarely.

The more marked the sclerosis, he states, the greater is the displacement of the vein. Normally, where an artery crosses a vein, at whatever angle, the line of vein at, and on each side of the crossing, is not even slightly diverted. In the earliest stages of sclerosis of the artery, the vein will remain unaltered, but will be hidden under the artery and for a narrow margin on each side, owing to the loss of translucency of the arterial wall. As the thickening of the arterial wall progresses the line of the vein comes to be diverted.

In a case of marked arterio-sclerosis, in which the vessels approach each other obliquely, the vein is at first diverted so as to lie alongside the artery, it then passes under it, and having reached the other side, again lies alongside the artery for a very short distance before continuing the former line.

In a case of very severe sclerosis, the vein though meeting the artery at an angle of say 15°, and leaving it at a similar angle, passes under it at right angles.

The same phenomenon may be observed where a vein crosses over an artery.

Evidence of obstruction of the venous flow.

One would expect that where a hard and thickened artery crosses a vein, obstruction to the blood in the latter would be caused, with consequent dilatation of the vein, a condition which has been described as "banking." As a matter of fact, Foster Moore says this is rarely seen, and my own experience as regards this is the same, though every now and then it is present beyond doubt, and is very striking when seen, though of all the cases I have examined in the past year, I can only recall three in which it was unmistakable.

The most marked case I ever remember was in a woman, aged

60, with a blood pressure of 240. The superior temporal artery crossed the superior temporal vein about one disc's breadth away from the disc. From the crossing to the disc the vein and artery were the same size, but beyond it the vein was quite twice the size of the artery, and very dark in colour. Elsewhere in the retina, where the arteries crossed the veins, there was pinching of them, but no evidence of interference with the circulation.

To discuss for a moment the importance of these various signs:

(1) Irregularity of calibre of the arteries. To me this is one of the commoner and more important signs of definite sclerosis, especially if one adds general diminution in the size of the artery as well. The normal ratio of artery to vein, according to Leber, is 2 to 3. In arterio-sclerosis I should put it at 2 to 4.

In following the course of the artery towards the periphery it can be seen to be narrowed for a short distance, and then to return to its original size; the variations are not gross, but quite unmistakable, and may be repeated several times in the course of the vessel.

One case I came across illustrates the importance of this sign. He was a man of 63, and his blood pressure was 210, and there was some albumen in the urine. There was no sheathing, no bright reflex, and no pinching of the veins, yet the arteries were small and showed definite variations in calibre.

This irregularity is not always present, but it is a most certain and valuable sign of high pressure when it is.

(2) Tortuosity of the arteries is one of the rarer and less valuable signs, and is one that is often seen as a physiological condition. When the small "corkscrew" vessels running towards the macular region are present, it is a corroborative sign, but I believe by no means so common as many of the others.

(3) Central light reflex exaggerated.

It is not only a bright reflex in the artery which is important; this is often seen physiologically. It is the hard sharply defined bright streak that indicates high blood pressure, and is a very important sign of this. At the same time I have been struck by several cases of very high blood pressure that showed practically no exaggeration of the reflex at all, other signs being present in abundance, as for instance in the case quoted above.

(4) Loss of translucency of the arterial wall.

This, when present, is again a very characteristic sign of severe sclerosis. You all know how the vein looks as if a gap had been taken right out of it, the artery apparently running through the middle of the gap, the explanation being that the vein is hidden by the thickened arterial wall which itself is not visible.

With one sign or another then, a hard artery is, as a rule, unmistakable. It looks hard. It is small in size and gives one the

impression of a round solid rod. I believe this to be due to a slight shadow, as it were, cast on the retina behind it, making it stand up in relief, and its reflex is usually, though not invariably, thin and bright, and its general colour pale.

(5) Where the artery crosses a vein.

1st. The vein is frequently pushed out of its direct course.

2nd. There is the appearance described by Foster Moore and quoted above. I have seen this, but must confess it had not impressed itself on my notice as very common.

3rd. There is loss of light reflex from the vein for a varying distance up and down from the arterial crossing.

4th. The vein appears to be pinched or indented; it is different from the gap appearance described above and there is no evidence of "banking;" it more resembles the condition of a rubber tube with a piece of string tied round it,

6th. Oedema of the retina—generalised oedema of the retina have not seen, but localized areas of oedema I have come across on two occasions only.

The congested appearance of the disc emphasized by de Schweinitz is seen in some cases, but is hardly as striking as the changes enumerated above.

In addition to these signs of arterio-sclerosis in the retina, Foster Moore[10] describes a condition to which he ascribes the title of arterio-sclerotic retinitis, and differentiates it from renal retinitis. In addition to the ordinary signs of sclerosis, retinal exudate is present in the form of small scattered white spots, irregularly circular in outline and variable in size, with no oedema or pigmentary disturbance, and the spots are usually scanty. Between the macula and disc is a common site for their occurrence, and sometimes a partial star figure is present at the macula. The white dots are slow to develop and slow to undergo changes, the dots may become confluent and may completely disappear and leave no trace. In addition, larger areas or small white plaques may be seen, usually in the central region. Pipe stem sheathing of the arteries may also develop. The changes occur very frequently in one eye only, though the ordinary signs of sclerosis are present in both eyes.

Pathology

Before passing to the consideration of the pathological changes that occur in the retinal vessels as a result of arterio-sclerosis, a few words must be said about their normal structure.

The central artery of the retina[11] from its entrance into the nerve until it reaches the lamina cribrosa has the structure of an ordinary small arterial twig. The intima consists of 3 layers: (1) elastic membrane; (2) a thin layer of subendothelial tissue; (3) endothelium.

When the lamina cribrosa is reached, the lumen becomes narrower, which must tend to cause eddies and so favour the occurrence of endarteritis and it may also tend to arrest foreign bodies circulating in the blood.

In addition certain structural alterations are observed.

The surrounding connective tissue becomes much denser, the elastic membrane is replaced by a feltwork of fine fibres, which separate the endothelium from the outer layers, and the subendothelial tissue seems to disappear. Therefore a retinal artery appears to consist of an endothelial lining and a connective tissue wall in which elastic and muscular fibres are present, but in the smallest twigs both elastic and muscular fibres disappear.

The central vein has a more delicate wall, consisting of connective tissue with curly elastic fibres, muscle cells are scanty, there is no true elastic membrane or subendothelial tissue, so that the endothelium rests directly on the connective tissue wall.

Within the eye there are no elastic elements in the walls of the retinal veins, which consist of endothelial lining and a connective tissue wall. Any or all of the structures may be diseased in arteriosclerosis, and the difference in structure between the retinal and central vessels produce differences in the microscopical lesions which are found.

In the central artery the intima is usually chiefly affected, and in the majority of cases all the lesions are inside the elastic membrane. The membrane may be thickened and itself split into two or more layers. Inside are layers of cells among which small curly elastic fibres are evident. These are often of some size and may form small elastic membranes within the true one, but they never reach all the way round. The lumen is usually lined by a single layer of well-formed endothelial cells, even when the layers beneath are extremely degenerate, which suggests that the essential lesion is not a proliferation of the endothelium, but of the subendothelial layers

Changes in the retinal vessels.—Disease of the intima takes the form of cellular proliferation but no elastic fibres are found.

Therefore, Coats concludes, the endothelium of arteries is incapable of forming elastic fibres, but that the subendothelial tissue, under pathological stimulation, is capable of laying down new deposits.

This subendothelial tissue being absent in the retinal vessels, no elastic membrane is normally laid down, and under pathological stimulation no new fibres are found in the intima.

In the retina the connective tissue wall is often greatly thickened and results in hyalin degeneration, and when the connective tissue wall thickens it encroaches more or less concentrically on the lumen, but when the intima is affected narrowing is always eccentric, the lumen being displaced to one side.

Coats concluded that white lines along the vessels are probably due to thickening of the connective tissue coat, whilst a simple narrowing of the lumen is due to endothelial proliferation. Disease of the intima is most probably due to a circulating toxin, whereas thickening of the connective tissue wall is probably compensatory for raised blood pressure.

The significance of changes in the retinal arteries

The retinal arteries, being end arteries, resemble the blood-vessels which supply the large ganglia of the brain, lesions of which lead to apoplexies (Knapp).[12]

Coats,[11] however, states that the ocular circulation must not be looked on as a mere expansion of the cerebral circulation, in the same sense as the nerve is of the brain. He states that it by no means always shares in disease of the brain, the angio-sclerotic processes being exceedingly irregular in their distribution, one region may be affected whilst another is normal.

Thus at the lamina cribrosa, which, as shown above, is a favourite site for endarteritic processes, the artery may be almost obliterated by a knob-like nodule and yet be practically normal a millimeter or two up the nerve. He says, therefore, that whilst disease of the retinal vessels is not a proof of disease of the cerebral circulation, it furnishes a presumption in favour of its existence. Conversely, absence of retinal change is no positive proof that other vascular areas are intact.

Again, fairly extensive disease of the retinal vessels may exist without ophthalmoscopic evidence ; but in spite of this, he concludes that they are the best indicator we possess as to the general state of the vessels.

Raehlman[13] in 210 cases of arterio-sclerosis found nearly half showed changes in the retinal vessels.

Hertel[12] says that a positive finding of sclerosis of retinal vessels assumes a similar condition of the cerebral vessels, but that the reverse does not apply.

Foster Moore[10], out of 44 cases of cerebral haemorrhage or thrombosis, found that 30 per cent. showed no evidence of disease in the retina; 27 per cent. mild or moderately severe disease; 43 per cent. severe disease and arterio-sclerotic retinitis.

Gunn quotes 17 cases of sudden onset of hemiplegia; out of which, 7 had definite retinal vascular disease, 3 slight vascular changes, and 7 showed no decided retinal changes.

Again, taking 46 cases of arterio-sclerosis Foster Moore found gross vascular lesions occurred in 21. In 25 cases there was no evidence of this; 18 of them were still alive. As a result he concludes that the relation between the condition of the retinal arteries and the arteries of the brain is both striking and close.

Finally, of the 156 cases of various retinal vascular lesions which[14] I attempted to trace to the end, out of 49 in whom the cause of death was definitely known, 22 died of cerebral haemorrhage or stroke, and possibly more, though the death certificates were too vague to be certain in all cases.

Group III

Chronic nephritis with secondary high pressure—arterio-sclerosis and heart changes.

In this group the primary lesion is in the kidney, associated with fibrosis, which leads secondarily to high blood pressure, sclerosis of the arteries and hypertrophy of the heart, as opposed to those cases which we have already discussed in Group II, which start with hyperpiesia, then develop arterio-sclerosis, eventually leading to sclerosis of the kidneys.

There are two important divisions of chronic nephritis, that of the infections and that of the intoxications, e.g., gout, lead, and alcohol.

(4) To diagnose between the primary nephritic and the primary arterio-sclerotic cases is not often difficult.

In the infectious group the history is generally distinct, the patients are younger and have not the vigour of the arterio-sclerotic cases, whilst lead and gout toxaemia when present is generally manifest.

The urinary changes differ from the arterio-sclerotic form in the persistent low specific gravity, lower nitrogen output, the more constant presence of albumen, and the persistence of granular casts.

The symptoms are more renal and cerebral in the nephritic, cardiac in the arterio-sclerotic.

Osler says that marked ocular changes are more common in the primary nephritic form and the retinal changes are degenerative, not simply haemorrhagic.

In the nephritic class remarkable variations are found in the arterial tension, persistently high in some cases, in others quite moderate, although associated with extreme arterio-sclerosis and hypertrophy of the left ventricle.

With regard to the changes found in the retina in these cases, two factors are responsible, the one vascular and the other toxic, and according as one or other of these predominates, so the retinal changes vary.[10]

In chronic parenchymatous nephritis, the vascular factor is much less prominent than the toxic. The blood pressure may or may not be much raised and the retinal changes are indicative of a toxic element. The picture one gets is as follows:

Numerous "cotton wool" patches scattered about, and associated with these much oedema, which may lead to retinal detachment. Retinal haemorrhages are not very plentiful, but may form a fringe

round the areas of exudate. The well known star shaped figure at the macula is not often seen.

A case that I saw recently of this type showed numerous cotton wool patches, much albumen in the urine, blood pressure of 200 mm. of Hg. and yet the arteries were only very slightly affected.

In chronic interstitial nephritis with which high blood pressure is always associated, the vascular factor predominates over the toxic, and the retinal vessels show signs of sclerosis, haemorrhages are more plentiful, and the star figure at the macula is frequently present. "Cotton wool" patches are much less plentiful and not so extensive as in the former variety.

In arterio-sclerosis, the retinitis which develops is probably, as Foster Moore[10] states, due to disease of the retinal vessels and corresponds with the vascular moiety of renal retinitis, but as the disease progresses the vascular disease in the kidney may so seriously involve the kidney substance, as to lead to renal insufficiency; so "cotton wool" patches, indications of this insufficiency, may appear in the retina.

Prognosis

The important question in these cases is not the prognosis of the eye condition; this is of secondary importance when compared with the prognosis as regards life.

Allbutt in speaking of hyperpiesia states that it may be cured or at any rate recur with less persistence, but if neglected ultimately leads to arterio-sclerosis, thus emphasizing the importance of early recognition of the condition if one is to be able to give a good prognosis.

Looking at it from the side of the general physician, Osler, with all other authorities, emphasizes the fact that the high pressure is not the disease, but a compensatory and salutary state, and in his experience the pressure may be kept at a reasonable height by treatment but that it never falls to normal again. In spite of this, however, a man may enjoy good health and do hard work with high pressure, whipcord arteries, and a hypertrophied heart.

Mackenzie[5] again found that some arterio-sclerotic patients lived for years, whilst others died of heart failure and apoplexy. These latter were always associated with gross evidence of disease of the heart, kidneys, and blood-vessels. In those who survived an ordinary life, there was no lesion of these organs more than one would expect from the age of the patients.

Therefore, in giving a prognosis, it is necessary to enquire into the condition of the kidneys, the state of the arteries, and the condition of the heart, with especial reference to its size and its functional efficiency.

James Taylor[15] also agrees that the prognosis depends, not so much on the actual appearance of the vessels as on the associated visceral changes, especially renal and cardiac.

In 1916[14] I undertook an inquiry into the prognosis *re* life of cases of vascular disease of the retina and found, as other observers have done, that the expectation of life varied enormously. To obtain a more accurate idea as to the prognosis, the cases were divided up according to their ages into decades and it was found that the age of onset made a great difference to the prognosis, it being much more serious in young subjects than in older ones, but the factor which made the greatest difference was the presence or absence of albumen in the urine, i.e., renal involvement.

The conclusions I drew from the study of these cases were, that retinal lesions were considerably more common in women than men, and that the cases were most numerous between the ages of 60 and 70, and next between 50 and 60. That the older the patient, the better the prognosis, as regards life, irrespective of the presence of albumen in the urine to a large extent. Whilst the younger the patient, the worse the prognosis, and especially so if albumen was present in the urine.

As regards the prognosis *re* vision. In many cases with haemorrhages the sight is not perceptibly affected as long as the macular region remains clear. In the grosser lesions, however, such as venous thrombosis, if the lesion is in the central vein, very little recovery is to be expected, and the eye may become glaucomatous. When only a branch is involved, it is often surprising how the sight will improve, but as the temporal vessels are almost always the ones involved, the macular usually suffers, but the sight may clear up eventually, leaving a vision of about 6/18 to 6/12, but the sight is patchy and uneven.

Treatment

This, I feel, hardly comes within my province to talk about; at the same time there are certain general lines which it is important for us, as ophthalmic surgeons, to know, as we are expected at times to indicate lines of treatment and give general advice to our patients.

The first and most important thing is to dispel anxiety and apprehension, as worry, nervousness, and mental strain increase the trouble.

Then, Osler's recommendations are worth remembering: A. A thorough study of each case is required; B. Determine the nature of the case, whether one of simple hypertension, arterio-sclerosis, chronic nephritis, or all combined; C. Reduce speed; D. Lessen intake, ordering a diet of low proteid content with large quantities of fluid; E. Elimination should be promoted in every way, by kidneys, bowels, and skin.

Next, it is important to remember that the high tension is not the disease, as Lauder Brunton, Mackenzie, and others have emphasized, nor is it a bad thing to be got rid of at all costs. As in most other diseases, it is most important to look round for possible sources of infection and toxaemia, the teeth should be carefully examined for pyorrhoea, and if present, proper treatment should be adopted.

Indigestion and the possibility of toxic absorption from the bowels must be most carefully gone into and constipation rigorously dealt with, if present, not only on account of the toxaemia caused, but also to avoid the harmful effects which may be produced by straining. This, I believe, is of the utmost importance.

Speaking generally, you want to place the patient under conditions giving the heart less work to do. After attending to the above, the question of diet next arises:

(1) Reduce the bulk of each meal.

(2) Reduce the proportions of those constituents of the foodstuffs which more particularly stimulate the cardio-vascular apparatus, e.g., various salts, soluble extractives, alcohol, etc.

(3) Encourage proper mastication[16] and insalivation, especially of soft amylaceous foodstuffs.

(4) Substitute as much as possible, boiled for roasted forms of fish, fowl, beef, and mutton.

(5) Reduce the proportion of animal and increase the amount of vegetable foodstuffs.

(6) Tea, coffee, and tobacco should be reduced.

Such exercise as the patient can undertake without distress should be encouraged, at the same time remembering that some of these arterio-sclerotic patients are extremely vigorous and need cautioning against undue exertion.

As regards the eyes, avoidance of stooping, straining, bending, and lifting is of importance, as such tend to increase extravasation of blood into the retina and cause fresh haemorrhages.

Ordinary care should be taken with light and print, but I see no reason for forbidding all close work, as is sometimes done.

Elsworth Smith[17] gives sound advice, I think, when he says: "If the conditions in the eye are so grave as to threaten the integrity of the organ, the patient should be placed in bed, but if the condition is not urgent enough to demand complete rest in bed, then the patient shall be urged to pursue the even tenor of his way, free as possible from all sources of worry and strain."

Such then are a few broad rules for the general management of the arterio-sclerotic patients that we meet. For further details of treatment, I must refer you to such works as Lauder Brunton, "Therapeutics of the Circulation"; Oliver, on "Blood Pressure"; Bodley Scott, on "Some Modern Remedies," etc.

Before leaving the subject of arterio-sclerosis and the retina, I must refer to the work of Thomson Henderson,[18] who devised an instrument for the purpose of measuring directly the height of the arterial diastolic pressure in the eye. His instrument is like the familiar Geneva lens measure, and is applied to the eye, usually through the lid, at its upper outer angle. He first showed that the intra-ocular fluid contents must lie at venous exit pressure, because this is the lowest attainable pressure of the intra-ocular circulation. The important point is, that the two pressures, venous exit and intra-ocular are in perfect equilibrium, so that if the intra-ocular pressure is raised by pressure of the fingers, the veins at their point of exit are constricted and the venous exit pressure is raised to the same level. The two thus rise together, and as the pressure is increased, it rises first to arterial diastolic pressure, and finally to arterial systolic pressure; when this point is reached the circulation ceases altogether.

By means of this instrument, we can obtain the exact index of the height that the diastolic pressure is above the intra-ocular pressure, or what is the same thing, the venous exit pressure. He puts it at 15 to 25 mm. of Hg.; that is to say, that the arterial diastolic pressure is only 15 to 25 mm. of Hg. above the intra-ocular or venous exit pressure. He has found it as high as 35 to 40 mm. of Hg.

Using the instrument, I have measured the diastolic pressure in several cases of normal and heightened general blood pressure, but I have not yet taken enough observations to come to very definite conclusions.

The pressure readings obtained with the instrument in eyes normal as regards tension, have varied from 9 m.m. of Hg. up to 17 mm., but to my surprise I have not found them vary appreciably with the height of the general blood pressure.

According to my results, the cases of highest general blood pressure, have not shown a corresponding rise in the pressure one obtains in the direct reading from the eye. I do not believe it is due to faulty technique, as this is not difficult if ordinary care is taken to see that the instrument is applied at right angles to the eye and besides, the results have been the same throughout. Nor is it due to an increased tension of the eyes, some were controlled by the tonometer and all the rest with the fingers, and they were normal as far as it was possible to judge. It is possible that a more direct relationship would have been found if the arterial diastolic pressure had been measured as well.

More recently Bailliart has introduced a new instrument, but, as far as I can see, the principle is the same as T. Henderson's, though the shape is different.

The method he recommends is as follows:

(1) Apply pressure until well marked pulsation is seen in the artery, then read off the force that has been employed, this gives the diastolic reading.

(2) Increase the pressure until the arterial pulsation is arrested. This gives the systolic reading.

(3) With the tonometer, take the height of the tension, whilst similar force is applied to the eye as in (1) and (2).

This gives the actual diastolic and systolic pressure in the retinal artery of that eye.

In normal eyes Bailliart found that the arterial blood pressure usually exceeds the chamber pressure by 5 to 10 mm. of Hg. during diastole, and by 30 to 35 mm. of Hg. during systole.

Assuming the normal tension of the eye to be 18 mm., this gives an average diastolic pressure of 25 mm. of Hg., and an average systolic of 50 mm.

Priestley Smith,[19] as the result of many experiments, places the average chamber pressure at 24 mm. of Hg. The central vein pressure, he states, is slightly higher than this as a rule, and he concludes that the exit pressure in the central vein is on the average not less than 25 mm. of Hg. The pressure in the central artery of the retina, at the point of entrance, he states to be probably from 90 to 100 mm. of Hg. The pressure in the retinal capillaries calculated from the relative cross sections of the arteries and veins, he concludes, is not far removed from 40 mm. of Hg.

These observations on the direct blood pressure in the eye should prove of great value when a little more is known about them, and their normal ratio to the general blood pressure understood, and I believe there is here a great field for further investigation.

We must now leave the subject of blood pressure and the retina and pass on to another question of some importance. That is the relation between glaucoma and blood pressure. Is there any connection between the two, or is high blood pressure a causative condition of glaucoma? At first sight it would seem natural to suppose that the higher the blood pressure the higher would the ocular tension be. Recent researches have, however, not borne out this supposition.

Craggs and Taylor[20] conducted a research into the relation between systemic blood pressure and raised intra-ocular pressure.

They compared the blood pressure readings in patients of the same age, with and without glaucoma. They found normal tension with very high blood pressure, and conversely high tension eyes with normal blood pressure.

In their glaucoma cases, the average systemic tension was not raised above the normal, and they came to the conclusion that arterial tension was very far from being a necessary factor in the causation of glaucoma.

Again, A. MacRae[21], after comparative measurements in glaucoma patients and controls, came to the conclusion that an altogether exaggerated importance had been given to the blood pressure as a factor in maintaining or increasing eye tension. He found that the blood pressure might alter greatly without any corresponding effect on the ocular tension being observable, and it was very doubtful if blood pressure was of any importance in the causation of glaucoma.

Foster Moore[22] in his paper on intra-ocular tension with especial reference to its variations in general and local vascular disease, says, "It is clear that such variations as do occur in the tension of the globe, are not caused by or associated with a like variation in general blood pressure," and he came to the conclusion that a rise in blood pressure, as a result of arterio-sclerosis, does not lead to a corresponding rise in intra-ocular pressure.

My own observations lead me to the same conclusion as the above. At the same time, everyone is familiar with the appearance of retinal vascular disease, that is sometimes seen after an acute glaucoma, which has been relieved by operation. Then, again, I have three cases, all occurring in painters, whose cases I would like to mention.

1. Iridocyclitis with secondary subacute glaucoma, haemorrhage in retina. Blood pressure 160, aged 46 years. Both eyes operated on.

2. Aged 48 years, subacute glaucoma right eye and left eye. Blood pressure 200, arteries thick in both. Retinal haemorrhages right eye. Both eyes operated on.

3. Aged 49 years, Haemorrhages in right eye. No albumen or sugar in urine, May, 1916. Still haemorrhages in retina and tension full, March, 1917. Tension higher October, 1917. Trephined January, 1918.

All these men were painters. They had no albumen and no signs of lead poisoning and yet they had definite signs of arterio-sclerosis and sub-acute glaucoma associated with disease of the retinal vessels.

In a different category and quite a different type of glaucoma associated with vascular disease, is the glaucoma which is so liable to follow on cases of venous thrombosis. As a rule, the cases of this type that I have observed, have been rather acute with vascular engorgement, but recently I have had two cases absolutely quiet and free from all signs of congestion, but with a steamy cornea, T.+2, and the sight abolished.

Finally, I believe there are some cases of apparently a low degree of chronic glaucoma with tensions about 30 mm. Hg. (Schiötz) in whom one finds high blood pressure and signs of arterio-sclerosis. Their glaucomatous symptoms and tension are to a certain extent relieved by attention to their general health and treatment directed

to the reduction of their blood pressure. Cridland has drawn attention to this point.

Thus there do seem to be certain cases in which there is a definite connection between intra-ocular pressure and intra-ocular vascular disease and raised blood pressure, but in the large majority of cases, there is, I believe, no direct relation between the two.

There is yet one other eye disease which in some cases is definitely associated with arterio-sclerosis and that is, a chronic form of iridocyclitis. I do not mean to say that it is caused by the actual height of the blood pressure, but whatever the toxin may be that is causing this, it probably also accounts for the iridocyclitis. I have seen several cases of this type and one of them, typical of the others, I will now quote:

Patient is a lady aged 56. Teeth clean, no gastro-intestinal symptoms or constipation, some rheumatic pains in joints, and knuckles of fingers are slightly enlarged. She developed a quiet iridocyclitis in August, 1918, first in one eye, followed by the other. The pupils have always dilated fully, but there was much "K.P." and fibrinous vitreous opacities which clear up and then recur for no apparent cause. In the course of examination, I found the arteries small, hard, and pale, no gross obstruction of veins, but slight indentation of them, and on measuring the blood pressure I found that it was 200 mm. of Hg. Since then, she has had occasionally a small striate retinal haemorrhage and a white spot or two of retinal exudate which slowly clear up. The inflammation obviously falls chiefly on the ciliary body as she has had no definite adhesions, but what she has had on several occasions, are the small cysts just on the free margin of the iris, which Doyne[23] described under the name of "Guttate Iritis" and which he attributed to gout, and these I have found in other cases of this type. At the same time none of these patients had any symptoms that one could say were definitely "gouty" and there was no albumen in the urine.

So called "central senile choroiditis" is another condition which is sometimes associated with high blood pressure, though it is a fact that one does not find sclerosis of the choroidal vessels associated with retinal or cerebral arterio-sclerosis, a fact pointed out by Russ Wood.[24]

The two following cases of this disease are rather striking:

(1) Blood pressure 270, arteries not grossly thickened but well marked central senile choroiditis; aged 79.

(2) Blood pressure 230, central choroiditis, no albumen or sugar, retinal arteries very small, variable in calibre, reflex not bright; aged 68. The condition closely resembled that known as "hole in the macula."

Nettleship[25] believed that central senile choroiditis resulted from

disease of the posterior ciliary arteries, and the two cases that I have quoted might well have been caused by this.

In now bringing my lecture to an end I do not wish you to think that I have exhausted the whole subject of the relation of arteriosclerosis to the eye. There is the question of optic atrophy and retrobulbar neuritis caused by pressure of the thickened wall of the carotid artery, and probably other complications that will occur to you. What I have endeavoured to do is to set forth the results of my own observations, at the same time quoting largely from recognized authorities on the subject, but what I did not wish to do was to copy out of a book all the facts that I could find and retail them to you.

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COLOUR BLINDNESS AND YOUNG'S HYPOTHESIS

BY

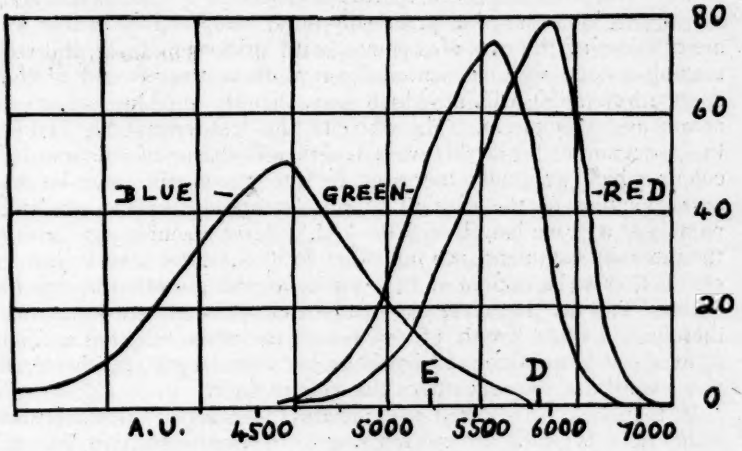
H. HARTRIDGE, M.D.

FELLOW OF KING'S COLLEGE, CAMBRIDGE.

(From the Physiological Laboratory, Cambridge.)

IT has been pointed out frequently as an objection to Young's hypothesis of vision that individuals who confuse red and green, and who, therefore, should be classed as either red blind or green blind, when they are told to describe a pure prismatic or diffraction spectrum, do not state that the end occupied by colours of long

wave length appears to them to be red (if green blind) or green (if red blind) as it might be supposed that they would, but on the contrary both classes are unanimous in describing it as yellow. This has been held to constitute not only a powerful objection to Young's hypothesis, but also to advance definite evidence in favour of Hering's views. Thus Dr. Pole, who was green blind, in describing his own case[1] says that one of the most forcible objections to Young's theory is "that it does not accord with the experiences of the colour blind. If there is one fact more unequivocally deducible from their evidence than another, it is that the less refrangible colour they perceive corresponds to the yellow and not to green. In my own case, which I believe is a typical one, my long wave colour is most vivid and positive, and it is an absolute certainty that its maximum splendour is excited by the buttercup, or by the pigment chrome yellow, or by the sodium line, whereas



objects that I hear called green give me no definite impression at all; sometimes they assume a debased, dirty or washed-out buttercup colour; sometimes they look black or grey, and sometimes they even give my opposite sensation, blue. How, therefore, it can be argued that my most brilliant buttercup sensation is excited by green objects rather than yellow ones is to me unintelligible."

These personal experiences thus clearly stated by Pole have been quoted by many authors[3] [4] [5] [6] as objections to Young's hypothesis of colour vision. Thus Edridge-Green says[2] "according to this theory (Young's) a colour blind with the red perceptions absent ought to see green as the most representative colour of the first unit, but the simple two unit (individuals) invariably say that

yellow is the colour which they see, and, as they are speaking of spectral light, there can be no question about the purity of the colour."

These facts, which have been held to discredit Young's hypothesis of colour, are, however, completely consistent with it.

In the above figure are reproduced Abney's equal area red, green, and blue sensation curves. These curves diagrammatically represent the facts that in the normal sighted the extreme less refrangible end of the spectrum to 6,600 A.U. arouses the red sensation alone. From 6600 A.U. to 5850 A.U., both red and green sensations are aroused in increasing amount, the former increasing less rapidly than the latter as the spectrum is passed through towards rays of shorter wave length. At 5850 A.U. a new variable is introduced by the rays beginning to excite the blue sensation. At 4850 A.U. the red sensation ceases to be affected, and at 4700 A.U. the green sensation suffers in a similar manner, leaving the blue sensation practically pure. Suppose now that we are considering the case of a person with green blindness, the red sensation alone will be excited by rays from the red end of the spectrum to 5850 A.U. at which wave length the blue sensation commences to come in. The whole of the less refrangible end of the spectrum as far as this wave length will appear of one uniform colour which gradually increases in intensity as the wave length corresponding to the summit of the sensation curve is reached, namely at a wave length of 6000 A.U. After reaching its zenith the intensity commences to fall till at 5850 A.U. the blue sensation commences to be excited and the purity of the sensation begins to suffer. The ray that most powerfully excites his red sensation has therefore a wave length of 6000 and its colour to the normal sighted is a pure yellow of slightly longer wave length and therefore very slightly more orange than the sodium lines.

If, therefore, he is asked to indicate the part of the spectrum which most typically arouses his long wave length sensation, he will point to a position in the spectrum near the sodium lines; if asked to name a pigment like it in colour, he will say "chrome yellow," and if a flower, he will say "the buttercup." The answers that he gives will be those of Dr. Pole. If now he were asked to say what colour the less refrangible end of the spectrum appeared to him, he would reply, the colour of the buttercup, of the sodium lines and of the pigment chrome yellow. But as persons with normal colour vision call these objects yellow, he also will do so, because he (like all of us) has learnt his colour names from them.

We may now ask him to examine the green part of the spectrum and to tell us what he sees. He will call rays of 5500 A.U. dirty buttercup, because, while they arouse his buttercup sensation strongly, they also stimulate his blue sensation as well; rays of

5100 A.U. he will call grey because his buttercup and blue sensations are approximately equally stimulated; while greens of shorter wave length still, he may call blue, because that sensation is the more strongly stimulated. In other words, he will name the green region of the spectrum as Dr. Pole names it; and he will name pigments in a similar manner according to the strength with which they stimulate his "buttercup" or his blue sensation.

Lastly, we may consider his statements concerning "whites" because it has been stated that since he is green blind and has only red and blue sensations he should call white "purple."

Now, he is in exactly the same position that persons with normal vision are, for he has learnt from infancy that certain pigments (e.g., zinc oxide), certain flowers (snowdrop), etc., are given the name "white"; but he finds that they arouse his blue and his "buttercup" sensation in certain proportions, and he therefore calls all other objects "white" which give him a similar sensation.

The statements made by an individual with green blindness are therefore those that would be expected on Young's Theory.

The case of the red blind may now be investigated in a similar manner. On examining the spectrum he finds that the red end is somewhat "shortened," and that the spectrum at this end has a uniform colour from its commencement to 5850 A.U. At this point and onwards the purity of his long wave length sensation is diminished owing to the stimulation of the blue sensation as well. If now he again examines the less refrangible end of the spectrum he finds that the intensity of the pure sensation that he receives increases rapidly as he passes from the end, till at 5850 A.U. it suddenly commences to be impure; if, therefore, he is asked to indicate the part of the spectrum which most vividly arouses his long wave length sensation, he will point to a position of 5850 A.U. near the Na lines. If asked to name a pigment like it in colour he will say "chrome yellow," and if a flower, "the buttercup." The answers that he gives are also those of Dr. Pole, and he, like Dr. Pole, will call his less refrangible sensation buttercup colour or yellow because he also has learnt his colour names from people with normal colour vision. The green region of the spectrum will appear dirty buttercup colour, grey, or bluish grey according to its wave length, as in the case of the green blind individual, green pigments also will be similarly classified. It should be noted that the neutral grey area of the spectrum is shorter in wave length than it is in the case of the green blind. White will be called "white" and not blue-green for a similar reason to that given above in the case of the green blind, namely, that the red blind have learnt from persons with normal vision to call objects white which arouse their green (buttercup) and blue sensations in a certain ratio.

Summary

On Young's hypothesis, a simple explanation can be advanced for the fact that both the red and the green blind call the less refrangible end (long wave length end) of the spectrum yellow, namely, that they have found by experience that their long wave length sensation is most strongly aroused by objects which the normal sighted call yellow. This also explains why they call greens, both spectral and pigmentary, dirty yellow, grey, or grey-blue according to shade. Also why they call white "white" and not purple, on the one hand, (green blind) or blue green, on the other (red blind).

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DR. EDRIDGE-GREEN'S THEORIES OF VISION

BY

J. HERBERT PARSONS,

LONDON.

DR. EDRIDGE-GREEN, in his many writings on colour vision and kindred topics, has advanced several theories. He has been indefatigable in his attempts to support these theories by statements based upon, and criticisms directed against, the work of other investigators, as well as by extensive series of observations of his own. It is not unnatural that in his enthusiastic advocacy of his views he should appear at times somewhat to underrate the work of his predecessors and contemporaries in the same scientific field. The sole object of the following remarks is in some measure to restore the balance. In my opinion, the time is not yet ripe for dogmatic allegiance to any of the many theories which have been propounded to explain the phenomena of colour vision. In my "Introduction to the Study of Colour Vision" (1915, Cambridge University Press) I have briefly described and criticized the chief theories, including Dr. Edridge-Green's. The value of a scientific theory depends not only upon its capacity to elicit useful generalizations from established facts, but also to stimulate the imagination to devise new methods of attack. In the latter respect is its utility chiefly to be judged, and therein lies the criterion which distinguishes it from vague speculations.

1.—The Function of the Rods

Dr. Edridge-Green says: "The rods are not perceptive elements." ("The Physiology of Vision," p. 134).

Some of the evidence in favour of the rods being perceptive elements is as follows:

1. *Comparative Anatomy.*—In most mammals, amphibia, and fishes, there are rods and cones, the former being the more numerous; in birds the cones are more numerous than the rods. In most reptiles (lizards, snakes, and tortoises) only cones are found. There are vertebrates possessing only rods, e.g., among fishes, rays, and dog-fish: among mammals, hedgehogs, bats, mole, and night ape (Nyctipithecus felinus). There are also animals of nocturnal habits possessing only rods. Owls, mice, and rell-mice have only a few rudimentary cones; rats also have a few cones.

The fact that there are seeing animals which possess only rods negatives Dr. Edridge-Green's statement. The fact that there are seeing animals which possess only cones proves that Dr. Edridge-Green's theory of the photochemical activation of the cones by visual purple secreted by the rods is not true for these animals.

2. *The Colourless Interval.*—If the eye is dark-adapted lights of low intensity from all parts of the spectrum, if seen at all, appear colourless. As the intensity is increased colour is seen, first for light of long wave-length (red) and at greater intensities for lights of shorter wave-length. The photo-chromatic interval is much diminished, if not, as held by many competent observers, entirely absent from the fovea. It is greater for the peripheral than for the paramacular region of the retina. It is probable, therefore, that the cones are insentitive to low intensities of light in the dark-adapted eye and are concerned, inter alia, specially with photopic or colour perceiving vision, and that the rods are specially sensitive to low intensities of light in the dark-adapted eye and are concerned specially with scotopic or achromatic vision.

3. *The Purkinje Phenomenon.*—With diminishing intensity of light and increasing dark adaptation the brightness of different coloured objects alters, the colours towards the red end of the spectrum becoming relatively darker, those towards the violet end brighter, so that finally the reds appear almost black and the blues white. In the colourless spectrum of light of low intensity seen by the dark-adapted eye the brightest part is at about 530 $\mu\mu$, i.e., the part which seen with higher intensities is green, and the blue end is sixteen times as bright as the red end. In the spectrum of higher intensity seen by the light-adapted eye the brightest part is at about 580 $\mu\mu$, i.e., in the yellow, and the red appears about ten times as bright as the blue. Although there is some difference of

opinion on the point, most competent observers (*e.g.*, König, Lummer, Nagel, v. Kries, and others), using the most refined methods, agree that Purkinje's phenomenon is absent at the fovea.

Purkinje's phenomenon strongly supports the view that two different mechanisms are at work in scotopic and photopic vision, the former being carried out by the rods, the latter by the cones (plus or minus the rods). The absence of the phenomenon at the fovea supports the view that the cones play no part in scotopic vision.

(4) *The Absorption of Monochromatic Light by the Visual Purple.*—The curve of absorption of monochromatic lights by the visual purple is almost identical with the luminosity curve of the achromatic scotopic spectrum, *e.g.* the point of greatest absorption is at about 530 $\mu\mu$, which is the brightest part of the colourless spectrum of low intensity as seen by the dark-adapted eye. Now the visual purple is found only in the rods. Hence it is justifiable to conclude that the rods and visual purple are specially concerned with scotopic vision.

(5) *Reversed Purkinje Effect.*—There can be no doubt that the visual purple is of fundamental importance in scotopic vision and that its accumulation is the basis of dark-adaptation. It follows that the relation of the achromatic scotopic luminosity values of two lights, *e.g.*, a green which is strongly absorbed and an orange which is weakly absorbed by the visual purple, should depend upon the concentration of the visual purple in the rods, *i.e.*, it should vary with the degree of dark adaptation. Stegmann has shown that such is the case. If a luminosity match is made between green and orange with intensities at which they appear colourless after 5-15 minutes' dark adaptation, and the lights are again compared after much more prolonged adaptation the orange appears much brighter and must be diminished to about three-quarters of its former intensity to restore the match.

(6) *Pupil Reactions and Electrical Responses.*—Sachs showed that the pupil-reactions vary with the luminosities of the lights, independently of their colour, both in the normal and the colour-blind. Abelsdorff confirmed these observations and showed further that maximum pupil-constriction occurs in nocturnal animals from green light, whereas in animals of diurnal habits the maximum is obtained from yellow light.

Similar, but more reliable and much more striking results, have been obtained from the electrical reactions, especially in day- and night-birds by Himstedth and Nagel, and Piper.

(7) *Photopic and Scotopic Colour Matches.*—The changes in the value of colour matches, formerly referred to as "deviations from Newton's law of colour mixtures," on altering the intensity of the light and the state of dark adaptation are explained by the theory

that the photopic values are cone-values, those at lower intensities rod- and cone-values mixed, and those at the lowest, rod-values. Thus, Albert showed that spectral yellow matched with a suitable mixture of red and green ceased to match on diminishing the intensity, and Ebbinghaus and Mrs. Ladd-Franklin showed that "three whites made by mixture of red and blue-green, yellow and blue, and green-yellow and violet darken unequally with proportionally diminishing intensity, the first least, the second more, and the third most." In general, matches valid for high intensities become invalid for low intensities and dark adaptation in the sense that the mixture which possesses the greater rod-value exhibits the greater luminosity in tone-free scotopic vision (v. Kries).

(8) *Visual Acuity.*—König, in 1897, made a very accurate series of observations on visual acuity with white and monochromatic lights. If the intensities of illumination are plotted as abscissae on a logarithmic scale and the visual acuities as ordinates the curve is a straight line of gentle slope up to 0.25 metre candle illumination. There is then a sharp bend and the curve becomes a straight line of much steeper slope up to the maximum. The same type of curve is seen with red, green, and blue light, but with red light the preliminary slowly rising part is very short. König himself drew the conclusion that two different elements of the percipient layer of the retina are involved, and that under low intensities of light vision is due to the activity of the rods, under higher intensities to that of the cones.

(9) *Critical Frequency.*—When two lights of different brightness, whether of the same or of different colours, are alternated flickering is observed. If the rate of alternation is gradually increased a "critical frequency" is ultimately reached at which the flickering disappears. The flicker phenomenon is dependent entirely upon the brightness of the lights and not upon their colour. T. C. Porter found that the relation between the intensity of illumination and the number of revolutions per minute at which a disc, half white and half black, must be run in order that the flicker may just disappear follows a definite mathematical law. If the rapidity of stimulation increases in arithmetical proportion, the intensity of illumination must increase in geometrical proportion in order that flicker may be avoided. Expressed in the form of an equation, $n = k \log I + k'$, where n is the number of revolutions, I, the intensity of the light, and k and k', two constants. This relation was found rigidly true for illuminations from 0·25 metre candle to 12,800 times this value. But the remarkable fact was elicited that below 0·25 metre candle, the constant k suddenly changed its value to about half its former value. These results have been confirmed by Ives, Dow, and many others. The change is exactly parallel to that found by König for visual acuity. It occurs at the same intensity, about 0·25 metre candle, and is almost universally attributed to the same cause, viz.,

that below 0·25 metre candle the rods are the percipient mechanism, above that value the cones.

(10) *Total Colour Blindness.*—There are cases of congenital total colour blindness, in which light is perceived but no colours. In these cases it is found that the brightness values of spectral lights correspond exactly with those of the normal dark-adapted eye stimulated by lights of low intensity. Comparison of the luminosity curves shows their almost complete identity. Whereas, however, the normal achromatic scotopic curve is valid only for low intensities of objective lights the achromatopic curve is valid for all intensities, though with the higher intensities photophobia occurs. The process of dark adaptation pursues the normal course in the totally colour blind. Exposure to bright light causes prolonged diminution of vision, and recovery follows the course which would be expected if it were dependent upon restoration of the visual purple.

All totally colour blind people have marked diminution of central visual acuity, and in many cases there is undoubtedly a central scotoma. The curve of visual acuity with gradually increasing light-intensities shows no sudden bend, as in normal-sighted people, but continues in the same direction as for lower intensities until photophobia sets in (König). The reduction in central vision tends to absence of binocular fixation and divergence of the optic axes. We may conclude that the foveal neuroepithelium, if it retains its function at all, approximates in character to that of the normal rods.

Uhthoff and von Kries have shown that the flicker phenomenon in the totally colour blind follows the same laws as in normal achromatic scotopia.

The simplest explanation of these cases is that cone vision is almost entirely, if not entirely, in abeyance, and that vision is carried out by means of the rods.

(11) *Night Blindness.*—Night blindness is found in several diseases of the eye, most commonly in retinitis pigmentosa and allied conditions, least adulterated by other pathological symptoms and signs in congenital night blindness. The last-named is congenital and hereditary.

In night blindness dark adaptation may be almost abolished or much slower than normal; in high degrees it is both slowed, and diminished quantitatively, i.e., the higher degrees of sensitiveness of the retina are never reached and the highest possible are reached only after prolonged exclusion of light from the eye. In low degrees central vision is normal in the light-adapted condition, and may be so in high degrees of the disease, e.g., in retinitis pigmentosa. Colour vision is normal with the exception of occasional diminution of sensibility for blue lights. In retinitis pigmentosa the field of vision is diminished, often almost down to the fixation point, and dark adaptation may be completely absent.

Purkinje's phenomenon is much less marked in the night blind. If a red and green are chosen, which are equal in luminosity for the normal and night blind in light adaptation, and the room is then darkened, the green then becomes much brighter than the red in the course of a few minutes for the normal, but only after a long time, if at all, for the night blind. Quantitative experiments show that for lights of short wave-length the increase in luminosity after after half-an-hour's dark adaptation is 10 to 100 times as great for the normal as for the night blind. With stimulation areas exceeding the foveal limits the sensibility for mixed white lights increases much more rapidly for the normal than for the night-blind on dark adaptation, but if both are tested with red lights there is very little difference. This is further proof of the diminution in the appreciation of Purkinje's phenomenon by the night blind.

The simplest explanation of these cases is that in them rod-vision is seriously disturbed or in abeyance. The pathology of retinitis pigmentosa supports this view. This disease is primarily due to vascular degeneration in the choroid, whereby the chorio-capillaris is destroyed. The pigment epithelium and the outer layers of the retina are dependent for their nutrition upon the integrity of the chorio-capillaris. From its proximity the pigment epithelium must suffer first. The production of visual purple in the rods is undoubtedly bound up in the integrity of the pigmented epithelial cells, so that primary disorder of the functions of the rods might be confidently anticipated on pathological-anatomical grounds. It is the rule in most nutritional disorders for the most highly differentiated and complex structures to suffer first. That there is good evidence of a survival of the cone-functions after destruction of rod-functions is evidence of an intermediary process such as that described.

12. *Anatomical Structure and Evolution.*—It seems almost inconceivable that two structures so similar in form and composition, in position and anatomical relations, should have such diverse functions as are attributed to them by Dr. Edridge-Green. Even in the human retina intermediate forms are seen of such a nature that it is difficult to determine whether they are really rods or cones, and in the fovea itself the cones are elongated and cylindrical, resembling rods more closely than elsewhere. Of the two the cones are the more complex in structure and the rods are absent at the most highly differentiated part of the retina. The natural conclusion, which is supported by analogy and by comparative anatomy, is that the rods are the more primitive visual element, and that the cones have become differentiated from them in the course of evolution. It is consistent with this view that the rods retain in man the primitive function of mere light perception, whereas the more highly differentiated cones have acquired the

more complex function of responding specifically to qualitative varieties of light stimulus. Yet they too retain the capacity to respond to the unanalysed light stimulus, a good example of what I have elsewhere in this Journal described as the apocritic principle of the evolution of perceptions.

Moreover, as Sherrington has pointed out, Dr. Edridge-Green's hypothesis as by the rods leaves unexplained their linkage to the retino-optic nerve-paths. It denies to one half—in some animals to much more than a half—of the optic nerve fibres the conduction of visual impulses. It makes difficult the attribution to these fibres of any afferent function.

Taken literally, Dr. Edridge-Green's statement that Nagel says "There is also not a single fact pointing to the view that the rods are percipient elements" is true. It is, however, misleading. The passage referred to is as follows:

"What has been said above (referring to the exact site of retinal stimulation) speaks only in favour of the cones, but not of the rods, as light-perceiving elements, and there is indeed nothing against the view that the latter remain passive in actual seeing, as amongst others was conjectured by Gad, who regarded the rods as elements concerned in reflexes. Two kinds of end apparatus of clearly different structure, which nevertheless possess the same function, must also give rise to doubts. These may, however, be regarded as eliminated, since the theory first propounded by M. Schultze and brought to the highest degree of probability by Parinaud and von Kries has rendered it possible to consider the rods as well as the cones as light-perceiving elements, and to account for the differences in structure of the two organs and the differences in their connection with the optic nerves by the suggestion of essentially different functions."

The arguments which have been gathered together above are the chief arguments in favour of the theory referred to by Nagel, generally known by the name given to it by von Kries, the Duplicity Theory. Like most generalizations of this kind it fails to account satisfactorily for all the facts. The phenomena of peripheral vision are difficult to reconcile with it, and Siven has collected other arguments against it. Dr. Edridge-Green, however, is probably the only worker in this field who denies *any* light-perceiving function to the rods.

II.—The Photo-Chemical Theory

Since Boll, in 1876, discovered that the visual purple was bleached by exposure to light, and more especially since Kühne's exhaustive investigations, a photo-chemical explanation of the action of light on the retina has been the most popular. Kühne found visual purple in the rods only, and this observation has been

confirmed and is generally accepted. Dr. Edridge-Green holds that the substance diffuses into the surrounding fluid and becomes distributed to every part of the outer layer of the retina. He found it between, and not in, the cones of the fovea. Hering has already suggested that visual purple may not be wholly absent from the cones, and the peculiar rod-like character of the human foveal cones makes it not improbable that they contain some of the substance. This would account for the (feeble) dark adaptation of the fovea. Dr. Edridge-Green has described in detail "currents seen in the field of vision not due to the circulation," and attributes them to movements of the fluid activated by the visual purple.

It does not *a priori* appear at all probable that vision depends upon the wide-spread diffusion of sensitized molecules in a fluid medium. If it were, it would be very difficult to understand how the extremely fine discrimination of minute objects, e.g., a visual acuity with a minimum visual angle considerably less than one minute, such as is often met with, could be obtained. It is far more probable that if the process is photo-chemical the reaction takes place in the neuro-epithelium itself. Possibly this is what Dr. Edridge-Green really means; but if so, it seems inconsistent with his explanation of the shifting and breaking up of after-images by shaking the head. It is surely unlikely, on physical grounds, that fluid filling the minute interstices between rods, cones, and pigment epithelium, should be shifted and scattered by merely shaking the head.

It is also improbable that the fovea, the part of the retina which is most sensitive in photopic vision, and which is specially differentiated for the discrimination of minute details, should be sensitised by fluid flowing along definite channels from the surrounding areas. The anatomical evidence in favour of such channels is of little value. Such as is derived from gross pathological lesions, like Sir Victor Horsley's case of choked disc with a macular star-figure in a case of intracranial tumour, is, in my opinion, valueless. This condition nearly resembles that found in albuminuric neuroretinitis; it is due to oedema of the retina, with abnormal exudates which so disturb the normal relations of the parts that evidence derived from it as to the normal structure is more than usually untrustworthy.

Burch, in 1913 (*Proc. Roy. Soc.*, B 86, p. 490), says, "Every hypothesis, whether mechanical, photo-chemical, or ionic, concerning the connection between the light-waves and the sensations they evoke, must of necessity rest ultimately on the theory of forced vibrations." Discussing the opto-chemical processes mathematically on this theory he derives four hypothetical colour-sensation curves, which meet the requirements of colour-mixtures and colour-

fatigue, on the same principle as the Young-Helmholtz theory, as modified by Burch himself. By altering the coefficient of friction he finds that the now flattened and extended curves explain the colourless scotopic spectrum. He then discusses the physiological elements of the visual sensation, and finds it necessary to hypothecate a "shunt-factor" whereby the strength of the sensation is governed. Otherwise "the theory of forced vibrations would indicate that with intense and long-continued illumination the several colour sensations must stand out more and more distinctly instead of becoming paler and tending towards white."

The theory of forced vibrations as applied to colour vision has been further elaborated in a paper by Houstoun (*Proc. Roy. Soc.*, A 92, 1916, p. 424). By making subsidiary assumptions, Houstoun, like Burch, is able to bring the theory into line with the Weber-Fechner Law, and also to explain the achromatic scotopic spectrum. He concludes that it is not necessary to assume two different mechanisms, and that "there is no decided evidence in favour of three classes of vibrators."

I am not competent to criticize the mathematics of forced vibrations. It appears that this theory supports Dr. Edridge-Green's theory of a single mechanism for both photopic and scotopic vision, if a further assumption is made. This assumption, if justified, must be pitted against the many arguments which I have adduced above in favour of two mechanisms.

Houstoun apparently does not find it necessary to resort to the shunt factor of Burch. Monochromatic light becomes whiter at high intensities because "when the impressed force on the vibrator is increased, the latter is more strongly damped, and hence the energy of the Fourier components of its motion more widely distributed over the spectrum."

While Houstoun considers that "the reception of light by the eye can be represented broadly by one class of vibrators," Burch's exposition shows that the three (or four) components theory is consistent with the theory of forced vibrations. On the principle, ***simplex sigillum veri***, Houstoun's view is the more acceptable, ***unless*** the phenomena are more easily explained by the more complex view. There is some reason to think that this is the case. For example, Houstoun finds it necessary to invoke Dr. Edridge-Green's theory that "the colour perceiving centre in the brain is not sufficiently developed to discriminate between the character of adjacent curves" in order to explain the apparent trichromatism of our ordinary sensations.

Moreover, as Burch (*Ophthalmoscope*, Vol. X, 1912, p. 57) pointed out in a criticism of Dr. Edridge-Green's view that the character of the stimulus differs according to the wave-length of the light to which it is due, and the discrimination of these differences is

a function of the brain, this hypothesis is directly opposed to the law of the specific energy of the senses. This law has been much discussed and is not universally accepted, but since Dr. Edridge-Green cites it with approval "(Physiology of Vision," p. 1) the argument should have some weight with him.

Probably most competent judges will agree that the light stimulus is of a photo-chemical nature and that the physical basis is one of molecular forced vibrations. They will, however, suspend judgment as to the exact details of the mechanism.

(To be continued).

ANNOTATIONS

Compulsory training and examination in Ophthalmology

During the past two years the General Medical Council has been urged to compel all medical students to study ophthalmology and to have them examined by specialists before they receive a licence to practise. Ophthalmic surgeons are so convinced of the necessity of this measure that the Oxford Ophthalmological Congress, the Council of British Ophthalmologists, and the Ophthalmological Society of the United Kingdom passed unanimous resolutions which have been considered by the General Medical Council. At the recent Congress of the Ophthalmological Society, the President, Mr. J. B. Story, P.R.C.S.I., devoted his opening address entirely to this question. He regretted that the General Medical Council had refused to institute a formal examination in diseases of the eye and had only consented to ask for some evidence of practical training in the subject. He pointed out that it was essential for every medical practitioner to possess a working knowledge of ophthalmology, and for two reasons: because rarely can a qualified man escape the necessity of treating eye patients at some time in his career, and secondly, the evidence afforded by an examination of the eye is needed for the diagnosis of many ordinary medical diseases; in fact, it has been stated that the ophthalmoscope is as essential as the stethoscope.

The General Medical Council had, he said, come to no logical conclusion, and had virtually tried to shift the responsibility on to the Hospital Medical Schools, who are empowered to refuse certificates to students who do not acquire a sufficient knowledge of eye diseases. The President reminded the Society that all the Irish licensing bodies had held a special clinical examination, and that this fact disposed of the objection that there was no time for

the student to learn the subject. He emphasized the value of an actual clinical test, for after thirty years' experience as an examiner, he knew how often candidates were well versed in book knowledge, but unable to diagnose the simplest ocular abnormality.

He said that the General Medical Council should be urged to institute the following reforms:

1. That each candidate for a licence to practise be compelled to attend an ophthalmic clinic for three months.
2. That the final examination shall include a clinical examination by ophthalmic surgeons.

A new Diploma in Ophthalmic Medicine and Surgery

A forward step has been taken by the London Conjoint Board in instituting a diploma in ophthalmic medicine and surgery (D.O.M.S., R.C.P. & S. Eng.). Candidates may enter for Part I of the examination, which includes the anatomy and embryology of the visual apparatus, the physiology of vision, and elementary optics, at any time after a registrable qualification has been obtained in medicine, surgery, and midwifery. Part II, upon optical defects of the eye, ophthalmic medicine and surgery, and pathology, may be taken on the completion of one year of special study of ophthalmology, and the production of certain certificates. The institution of this diploma amply justifies the efforts of those who have advocated the adequate study of ophthalmology and furnishes the best answer to the attitude of the General Medical Council towards the matter. We congratulate the Royal Colleges upon their enlightened action.

Artificial Daylight

The problem of artificial daylight is one of considerable importance in many industries. One of the recent attempts at solving the problem consists in the use of carefully combined colour reflectors in contrast to the earlier methods in which transmission through tinted screens was employed. The apparatus is known as the Sheringham daylight and is the result of the combined work of a physicist, Mr. L. C. Martin, an artist, Mr. G. Sheringham, and an expert on camouflage problems, Major Klein. Mr. Martin read a paper on the light before the Illuminating Engineering Society. The paper and the subsequent interesting discussion are fully reported in the *Illuminating Engineer* for February, 1920. One very interesting point was the difficulty of defining daylight on account of its great variability. In an editorial in the same journal the editor writes: "In order that the whole subject may

be placed upon a scientific footing, it appears essential that a standard of 'Normal' daylight should be defined, and the degree of accuracy with which 'Artificial daylight' units for various purposes should approach this standard specified." He also points out the necessity for a standard white surface and a standard nomenclature of colour-tints, for which latter the list prepared by the Société des Chrysanthémistes in France might serve as a basis.

In the subsequent discussion a communication from the United States by Mr. M. Luckiesh showed the extensive use that is being made of artificial daylight in industry in that country. The source of light described by him are tungsten (Mazda C) lamps equipped with coloured glass filters.

ABSTRACTS

I.—TETANUS FOLLOWING OCULAR LESIONS

Castelain and Lafargue.—On tetanus consecutive to ocular lesions. (Du tétanos consécutif aux lésions oculaires.) *Annal. d'Oculist.*, Vol. CLVII, January, 1920.

Castelain and **Lafargue**, having found a case of tetanus after eye injury, give notes of it, together with a considerably detailed review of the features of the published cases. The soldier reached their ambulance with a diagnosis of traumatic conjunctivitis, but on arrival showed panophthalmitis. As it was the rule to give anti-tetanic serum at once, it was taken for granted that such had been administered, but no positive note was made that he had had serum as a prophylactic. While he was striking a horse's shoe in the cold state, on November 25, 1916, a chip flew up and struck his left eye, a fragment of metal lodging in the upper part of the globe; thirty-six hours afterwards he presented exophthalmos, chemosis, oedema of lids, yellowish opacity of cornea—panophthalmitis of a rapid character, calling for chloroform and exenteration of left and extraction of the foreign body on the 27th. Apart from some chemosis there were no untoward signs till the seventh day, when, in the presence of swelling of the face, of difficulty in opening his mouth, of the symptoms noted by the patient, who said his teeth were on edge, Dr. Darrieux at once thought of cephalic tetanus and started the necessary treatment. On the eighth day the diagnosis was confirmed by still more defined symptoms, viz., complete paralysis of the whole of the left seventh nerve, contracture of the right face, difficulty in opening the mouth—trismus, ophthalmoplegia of the right. He presented the aspect described by Courtois-Suffit: he had loss of

power of muscles of the right eye, drooping of lid hiding most of cornea, and the wounded man, appearing asleep, threw his head back to see out beneath the drooping lid. The forehead was wrinkled, the eyebrow raised. With the lid raised the gaze was altered, the paralysis of the muscles no longer allowing the eye to fix near objects, only a slight outward movement remaining. It was not possible to say whether the left muscles were also paralysed, the chemosis obscuring the movements of the stump. Right pupil in extreme miosis and inactive to light, not possible to say whether any reaction on accommodation. In the present case the facial palsy was the first symptom, the trismus the second, and the ophthalmoplegia appeared later, but all appeared within twelve hours. Towards the twentieth day the muscles of the right eye did not seem more than paresed, the facial contracture was perhaps less marked, and the trismus less intense. In the following days the improvement became more marked, so that the ophthalmoplegia was cured about the forty-fifth day, the facial palsy disappearing some days later.

Although a bacteriological examination was not done, the authors consider the diagnosis of cephalic tetanus seems certain; in the photograph reproduced the risus sardonicus can be seen, profound apathy being shown on the one side of the face and a terrifying frenzy on the other. In the motor oculi paralysis and abducens paresis, the facial palsy and the trismus, the slowness of pulse, the acceleration of respiration, lies evidence that the toxin had affected the nuclei of the third, sixth, seventh, and tenth cranial nerves. Apparently a bulbar intoxication, and the work of Guillain and Laroche, who have studied the experimental fixation of the tetanic toxin on the masticator nucleus, would be a new proof in favour of this pathogenic theory. Absence of other than cephalic signs in this case may be due to the early and vigorous treatment used, for in many of the published cases time had been lost waiting for the diagnosis to be clinched. Antitoxic and antispasmodic treatment were both used in this case, the former by massive doses, 60 cc. daily, of antitetanic serum given intravenously in the hope of neutralizing the toxin produced, and circulating in the blood (although not operative on the toxin fixed in the nerve elements), the latter by injections subcutaneously of carbolic acid. Employed alone, 6 gramme doses of chloral were without effect, while the carbolic solution showed clearly its efficacy, for the stoppage of it in the course of the disease, from the 7th to the 13th December, was followed by the reappearance of the crises, which ceased on resumption of the carbolic, December 16th. These subcutaneous injections are painless and without reaction; they have no harmful effect on liver or kidney, since the albumen and the sugar that appeared at the start of the illness disappeared in spite of the

continuation of the treatment. Daily he got 1.2 grm. of carbolic acid, *i.e.*, 13.5 grm. in all, without altering the colour of the urine. Of the anti-tetanic serum he had 900 cc. intravenously in 34 days without incident, so that the efficacy and innocuousness of the massive doses of serum and carbolic acid used jointly are well established in this case.

In the 1916 *Annales d'Oculistique*, Schneider stated that in the literature he had found only 19 cases. Add to these Vinsonneau's case, and Rollet's, quoted by Goëtz in his Lyons thesis, 1916, that makes 21 and the present one brings the grand total published to date up to 22 cases. (The authors have apparently missed Sattler's [Cincinnati] case published in the *Trans. Amer. Ophth. Soc.*, Vol. XV (1917), p. 234, abstracted in BRITISH JOURNAL OF OPHTHALMOLOGY for March, 1919, p. 124).

Four full-page tables are given with the following points in the published cases, author, cause, primary lesion and its complications, date of appearance of the first sign of tetanus, operation done, final result.

In nine cases the injury was the stroke of a whip, in three gunshot, in eight by objects presumably contaminated by earth contact, viz., pitchfork, arrow, reed grass, chip of iron, chip of glass—an earthy or equine origin is thus evident in the majority of cases. In 18 cases the primary lesion is indicated, in 13 wounded there have been corneal lesions, of these 10 were penetrating, no details in the other three; also three penetrating wounds of globe, two of sclerotic, and 1 (Vinsonneau's) of conjunctiva. Apart from the iritis as noted by Rust in his case, the most common complication has been panophthalmitis—18 times, suggesting that panophthalmitis is a necessary condition for the bacillus of Nicolaier. One is also struck by the rapidity of the appearance of this complication, since in 12 cases where this point was noted we have three cases on the second day after the injury, six on the third, one each on the fourth, sixth, and eighth day. Apart from the cases of Dénucé and of Mauch, in which details are wanting, the period of incubation appears to be most often six to seven days, but in the Ramiro-Guedes case trismus appeared on the third day, and in Schneider's tetanus first showed on the 13th day. The evolution is usually the same—towards the sixth or seventh day after the accident one notes trismus, difficulty in swallowing, and facial paralysis on the same side as the lesion, i.e., cephalic tetanus, the syndrome of Rose. In 17 cases where the order of appearance of the symptoms is noted, trismus is generally the first, facial palsy appearing shortly after, preceding it in only three cases. Ocular palsies were noted in six cases, either on the side of the lesion or on the other, and either bilateral at the onset or only later, the motor oculi being the affected nerve, partially or completely. In Schnitzler's and the present case paresis

of the sixth nerve was also present. Generally the tetanus spreads beyond the cephalic type of Rose; about three days after these signs appear, the generalized nature of the affection is evidenced by spasms of the limbs and unfortunately in too many cases, 18 in the series, death ensues, the prognosis being worse the earlier the onset. In the successful cases the cure was reached in about one month; of four cured cases three had had exenteration while all the 10 enucleated cases died. Other factors have to be considered, but all the non-fatal cases were operated on early, second, third, and fourth day, while one exenteration done on the fifteenth day died, as also enucleation cases done on the fourth, fifth, sixth, eighth, eleventh, eleventh, fourteenth days. In half the cases no note is made as to the treatment given, but in six cases where it says serum was injected no note is made of quantity, channel, or date. Several of these cases died so soon that no conclusion can be drawn as to the value of this treatment.

The authors give the following guiding principles; in penetrating eye injuries from gunshot, pieces of glass, and chips of metal, in which latter the metal may have had contact with the soil of the workshop, one should carry out, always and as early as possible, an injection of 20-30 cc. of anti-tetanic serum; secondly, if by the second or third day there is any appearance of panophthalmitis, perform exenteration at once and frequently irrigate the socket with peroxide; thirdly, if, in spite of these preventive means, one finds later trismus, difficulty in swallowing, and facial paralysis, it is necessary to make forthwith daily intravenous injections of 60 cc. of serum and simultaneously subcutaneous injections of the same quantity of 2 per cent. carbolic acid, which appears to have an analgesic and antispasmodic effect. 15-20 days of such treatment may reasonably be expected to prevent generalized tetanus and gradually lead the way to a cure.

W. C. SOUTER.

II.—EYE LESIONS DUE TO POISONOUS GASES

(1) **Rathnakar, R. P. (Manchester).—A case of nystagmus caused by mustard gas.** *Lancet*, March 15, 1919.

(1) **Rathnakar** reports from Dr. Hill Griffith's clinic the case of a soldier, a signaller, who, after being gassed, developed nystagmus. Griffith had previously seen two other cases, but, being sceptical as to their freedom from nystagmus prior to the gassing, took no particular note. In the present case Griffith seems to have been satisfied that the patient had not previously suffered from nystagmus, since, previous to joining up, he had been a microphotographer at

one of the largest London hospitals and had had excellent vision. The nystagmus was of the lateral variety, constantly present, but more marked on extreme outward and inward movement. Fundi normal. Retinoscopy under atropin + 1D. V.A. 6/18 partly, not improved by glasses. Fields normal. Three months later the condition was unchanged.

ERNEST THOMSON.

(2) **Derby, George S. (Boston).—Ocular manifestations following exposure to various types of poisonous gases.** *Trans. Amer. Ophthal. Soc.*, Vol. XVII (1919), p. 90.

(2) The value of **Derby's** communication is enhanced by the fact that it is illustrated by six coloured and three black and white pictures of the lesions of the eye produced by poisonous gases, reproduced by the courtesy of the Medical Research Committee of the British War Office. Among the gases first (1915) used by the Germans the so-called "lacrimatory gas" did not cause more than temporary lesions of the conjunctiva, which even in the more severe cases usually recovered within two weeks. Much more serious were the injuries produced by mustard gas (dichlorethyl-sulphide), first used extensively at Ypres in 1917. It caused, after a delay of a few hours (two to six), painful irritation of the eyes and sometimes vomiting, and in those severely affected (about 20 per cent. of the whole) considerable redness and swelling and the formation of bullae of the skin of the eyelids. The conjunctiva was markedly injected, and might show a considerable degree of chemosis, especially in the culs-de-sac, upper and lower. The region of the palpebral fissure was most severely affected, and in badly burned cases there was often an area of solid white oedema in the palpebral aperture, which presented a very striking and characteristic appearance. The corneal lesions varied in severity. In the milder cases there was a very slight roughening of the corneal epithelium, which might or might not stain with fluorescein. In moderately severe cases there was a marked roughening of the epithelium, with irregular greyish areas of opacity scattered throughout the cornea, an appearance likened by the British to that of an orange skin. In the most severe cases a saturated grey band, sometimes of almost porcelain-like whiteness, traversed the cornea in the region of the palpebral fissure. Secondary infection might result in ulceration of the cornea or later even in panophthalmitis and loss of the eye. But fortunately the last-named condition was rare. The British observed only about ten cases of corneal ulceration which impaired the sight of the eye. Teulières saw among 1,500 cases one instance of panophthalmitis and three instances of ulceration of the cornea. The lesions of mustard gas are in the nature of a chemical burn of the tissues of

the eye. Pissarello has exceptionally seen cicatricial ectropion follow burns from mustard gas. Sir William Lister states that occasionally the actual liquid (the gas in its original form is fluid) from the shell in which it is contained passes into an eye, and then produces results similar to those caused by other liquid caustics. It is unlikely that disease of the posterior half of the eyeball, as neuro-retinitis, follows exposure to mustard gas.

Pathology

The Americans had no provision up to the time of the Armistice for the pathological examination of gassed eyes, so that Derby quotes from one of Sir William Lister's reports dealing with the matter. From that report it appears that such eyes showed changes in the corneal epithelium, the substantia propria, and the conjunctiva. The changes in the corneal epithelium included diminution in the thickness or actual exfoliation and exposure of Bowman's membrane. In some cases the substantia propria showed an infiltration with round cells between its layers. In the ocular conjunctiva granular masses were present in the subepithelial tissue, which probably corresponded to the solid white exudate mentioned before.

Experimental Work

The communication includes an account of important experimental work carried out by Warthin and others, chiefly upon dogs and rabbits, respecting the effects of mustard gas upon the eyes. These experiments were of two kinds: (1) Mustard gas was applied with a fine pipette directly to the centre of the cornea in uniform minute droplets, and (2) animals were exposed to the vapour of the gas. Fifteen minutes after the gas was applied congestion of the upper palpebral conjunctiva was noted, and in one hour the conjunctiva had become oedematous, until, finally, there was a rim of swollen conjunctiva about the cornea. Corneal changes occurred in five or six hours, and consisted in faint clouding, roughening, and irregularity; in eight hours the cornea was porcelain-white and there were irregular, rounded areas devoid of epithelium. The denuded area of the cornea increased in size, and the lower half often showed an opaque band running horizontally. Pus in the anterior chamber was sometimes met with. During the second week the corneal opacity remained without alteration, but the exudate decreased somewhat. During the third week a sluggish reparative process began. Ectropion of the lower lid was noted. Warthin found that changes varied enormously with the presence or absence of treatment, especially by a one-half of a 1 per cent. solution of dichloramine—T in chlorcosane. If no treatment was adopted purulent panophthalmitis often developed within three

weeks, and the eyes were destroyed within six weeks. The panophthalmitis was due entirely to secondary infection. Microscopically, the earliest changes in the cornea consisted of pyknosis, contraction of the epithelium and of the substantia propria, most marked at the corneal vertex, extending down to Descemet's membrane. This was followed by a loss of nuclei, until by the twelfth hour the apex of the cornea showed complete necrosis. Desquamation of the epithelium and polynuclear infiltration of the sclero-corneal junction began in about five hours. The earliest signs of regeneration of the substantia propria of the cornea were noted at the end of 65 hours, and continued for several weeks, with development of a highly vascularized corneal cicatrix. The severity of the corneal lesion was in direct proportion to the concentration of the gas and the period of exposure. As the result of secondary infection, iritis and irido-cyclitis were common at about the third to sixth week, but they occurred only in the uncared-for cases. Meyer (who discovered mustard gas) stated that by its injection he had produced a metastatic conjunctivitis, but in eight animals Warthin failed to verify this conclusion. Great differences have been observed in individual susceptibility. Marshall, Lynch, Smith and Williams, of the Medical Division of the Chemical Warfare Service, found that as regards the skin, 20 per cent. to 40 per cent. of the white men tested showed a certain degree of resistance, while 78 per cent. of the negroes tested, manifested a similar resistance. Individual differences in sensitivity were also noted in experimental animals. It is suggested that hypersensitivity to mustard gas may occur in those who show the picture of a persistent thymus and characteristic hyperplasia.

Treatment

The eyes should be washed out after exposure to gas with a 1 per cent. solution of sodium bicarbonate and later with a lukewarm solution of boric acid, salt, or sodium bicarbonate, and this should be followed by the instillation of a drop of oil, of which the best was found to be liquid albolene. The eye should never be bandaged. When there is much blepharospasm and irritation, atropin (1 per cent. or stronger) should be used, and the patient be supplied with a shade or a pair of dark glasses. When secondary infection takes place a weak antiseptic solution, as argyrol or protargol, was found to be of benefit. Septic ulcers of the cornea should be treated as in civil life. The patient should be sent outdoors as soon as may be and given light duty, mainly with a view to combat the possibility of the beginning of a neurasthenic tendency. Cocain should be banned.

S. S.

III.—MIXED TUMOURS OF THE LACRIMAL GLAND

Birch-Hirschfeld, A. (Königsburg).—A contribution to the study of mixed tumours of the lacrimal gland. (Zur Kenntniss der Mischtumoren der Tränendrüse.) *Arch. f. Ophthal.*, Vol. XC, 1915.

Birch-Hirschfeld describes a case of this kind in a man 57 years of age who had observed for four years a prominence of one eye. There was marked displacement of the globe forwards and downwards, total absence of movement upwards and outwards, diminution of vision, hypermetropic astigmatism, and slight hyperaemia of the optic disc. He removed the tumour by Krönlein's operation. After a few months the position of the eye, its movement and vision were completely restored, while the astigmatism caused by the pressure of the growth on the globe had disappeared. Six years after the operation there was no sign of recurrence of the tumour or metastasis. The new growth was examined microscopically, and his findings are described in detail.

A study of this case led the author to make a critical survey of similar cases so far as the description of the different writers justified, in his opinion, their inclusion in this class—he collected 75 cases recorded in the literature on the subject to which he had access—and the result of this review is embodied in the following conclusions:

1. A considerable number of the new growths found in the lacrimal gland and its immediate neighbourhood, and described under a great variety of names may be grouped together in one class under the general term of "mixed tumours."

Birch-Hirschfeld comments on the frequent difficulty, or even impossibility, of determining the exact point of origin of these new growths, and hence believes that many cases have been recorded as tumours of the lacrimal gland which had no connection whatever with it, while others, true cases, have been reported merely as tumours of the orbit.

2. Histologically these tumours are distinguished by their very complex structure. They contain cells of epithelial character arranged in the form of glandular lumina, or reticular bands, or solid processes. Morphologically these cells are in parts absolutely identical with true epithelium; a basement membrane, typical intercellular bridges, and sometimes, too, signs of cornification can be observed. The variability in the relation of these parenchyma cells to the surrounding stroma, which at one time shows myxomatous or hyaline degeneration and at another contains islands of cartilage, produces a picture that in many places closely resembles an endothelioma, in others typical cylindroma, in other parts again adeno- or chondro- myxoma.

3. The view of the epithelial character of the parenchyma is a more probable one than that of their endothelial origin; and the author has been led to this conclusion in great measure by the study of mixed tumours of the salivary glands to which the lacrymal growths bear a very close resemblance.

It is this complex structure with the varying arrangement and shape of the cells, the changes of the stroma, and the variation in the relative preponderance of cells or stroma, that has given rise to the great diversity in the reports of different writers and to the bewildering confusion in the terminology of these growths. The question of the origin of the cells is one on which there is great difference of opinion, a similar difference being found among pathologists with regard to tumours of the salivary glands. It may be noted that Parsons ("Pathology of the Eye," Vol. II, p. 752) distinctly favours the endothelial origin of the parenchyma, and regards these neoplasms as almost identical with the mixed growths of the salivary glands, which are generally held to be endothelial: he pertinently points to the occurrence of cartilage in the tumours as difficult of explanation on the theory of an epiblastic origin. Birch-Hirschfeld, however, as stated, takes the opposite view, and according to him there is, on the Continent at least, a considerable weight of opinion in favour of their epithelial character.

4. Clinically, these tumours are at first of a very slow growth, but they may suddenly take on a much more rapid development and become malignant, inasmuch as they may lead to local recurrence and the formation of metastatic growths.

5. The early and complete removal of the tumour is therefore indicated, and this should be carried out as far as possible with the help of blunt instruments.

6. As regards the origin of these mixed tumours in the lacrimal and salivary glands, it is very probable that they are due to a displacement of the embryonic tissue through some disturbance in the process of development (possibly in the third month of foetal life, when—in the case of the lacrimal gland—the embryonic cells come into relation with the primitive cartilage of the frontal bone).

THOMAS SNOWBALL

BOOK NOTICES

(1) Une Échelle de Clarté. (2) Moyens de Contrôle de Verres de Lunettes et de Systèmes Optiques en général. (3) La Théorie de Gauss. By M. TSCHERNING. (*Mathematiskfysiske Meddelelser*, 1918-19.)

These are three interesting papers by the well known professor

at Copenhagen of which here only a brief and inadequate notice can be given.

(1) *A Scale of Brightness.*—The eye can easily perceive at the same time two objects, when the intensity of the illumination of one is many thousand times greater than that of the other. According to Fechner's law (in the simple form in which he first gave it) the sensation of brightness will vary as the logarithm of the stimulus with the addition of a constant. Tscherning adopts this method, in a somewhat similar manner to that universally used by astronomers in scaling stellar magnitudes, or stellar brightness. He takes as his physiological unit the minimum light just visible on a dark background when seen by a normal eye adapted to light. This he finds by calculation to be the brightness due to the illumination of a sheet of white paper which is illuminated by a candle at the distance of 45 metres. If the illumination is 1,000 times greater (10^3 greater) he would call its brightness 3, *i.e.*, three physiological units. Unfortunately, Fechner's law as first stated does not hold for very low illuminations, so one ought not to press

S.	Distance of the Candle.	c.	S.	Distance of the Candle.	c.
$\frac{6}{6}$	0·29 m.	4·4	$\frac{6}{24}$	1·87 m.	2·8
$\frac{6}{9}$	0·77 m.	3·5	$\frac{6}{36}$	2·10 m.	2·7
$\frac{6}{12}$	1·14 m.	3·2	$\frac{6}{60}$	3·00 m.	2·3
$\frac{6}{18}$	1·51 m.	2·9			

it too far. However, as the light minimum varies so enormously with the condition of the eye, and one must form a unit of some kind, it is idle to raise objections to his unit, especially as it is found that it is nearly that of a VIth magnitude star which is about the extreme limit of visibility to most observers.

The apparatus he has devised for testing purposes is very ingenious. With an arrangement of two parallel blackened glass plates, one of which can be moved, the image of a standard light can be seen after 2, 4, 6, etc., reflections. As about 4 per cent. of the incident light is reflected, the illumination decreases to 0.0016 after two reflections, to 0.00000256 after four reflections, and so on. A table he gives of the relation between visual acuity and the illumination of the test types is interesting. The symbol *c* denotes the author's scale of brightness in his physiological units.

One sees that a good illumination is required in order that the standard visual acuity may be obtained by a normal person. One

candle at a distance of 29 cm. is about equivalent to the illumination of 12 candles at a distance of one metre.

(2) *Methods of testing Spectacle Lenses, etc.*—After first describing the usual method of neutralization, he points out that this method fails when dealing with lenses of high power and with menisci. As is well known, the concave glasses in our test cases are numbered according to their real dioptric value, and the convex glasses are numbered so that when held in contact with a concave glass of the same number neutralization will occur. The actual value of convex glasses is rather less than their nominal value, but the difference is slight except with thick convex glasses of high power. For instance, suppose a + 20 D (nominal) be of thickness 7·8 mm. and refractive index 1·523, although it is exactly neutralized by a concave of − 20 D, its actual power will only be + 18·56 D.

On p. 5 there is a misprint; the fraction should be inverted, and one should read $F = \frac{-F_1 F_2}{F_1 - F_2 - d}$

With menisci a serious error will arise if a meniscus of the same strength as the correcting glass from our test case be given. Suppose that − 15 D is found to correct the error, a much weaker meniscus lens will be required for the spectacle lens, as its effective strength is greater in that case than its real strength. This results from the fact that the principal points of a concave meniscus lie outside the glass towards its concave aspect. Attention was drawn to this point in a paper on Periscopic Lenses (*Ophthalmoscope*, July, 1914). The method of neutralization fails with menisci, both for this reason and also because the surface of the meniscus will only touch the neutralizing lens in one point.

The author's instrument is too complicated to describe, but it would seem to be extremely efficient and easy to use for determining the real optical power of any lens, whether the incident light be axial or eccentric.

(3) *Gauss's Theory.*—This paper is purely mathematical, and shows that Gauss's method may be applied not only to axial incidence but also to eccentric incidence. A geometrical device is also given by which the position of the focal lines may be found. The paper must be read by those who are interested in the subject; it is impossible to do it justice in a brief notice. Professor R. A. Sampson published some work on the same subject about twenty years ago, but Tscherning's treatment is much simpler in this paper.

A. S. PERCIVAL.

NOTES

Honour.

CONGRATULATIONS to Mr. Arnold Lawson, whose name figured in the King's recent birthday list of honours as having had conferred upon him the distinction of a Knight Commander of the Order of the British Empire.

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Appointments

HARRY LEE has been appointed specialist medical referee under the Workmen's Compensation Act, 1906, for County Court Circuits, Nos. 12 and 14, with a view to his being employed in ophthalmic cases.

J. Burdon-Cooper has been appointed lecturer in physiological optics to the Department of Ophthalmology in the University of Oxford.

Humphrey Neame has been appointed assistant surgeon to the Central London Ophthalmic Hospital.

J. C. W. Halliday has been appointed ophthalmic surgeon; E. A. Brearley, R. G. Waddy, and H. H. Rutherford assistant ophthalmic surgeons to the Royal Prince Alfred Hospital, Sydney. Percy S. Webster has been appointed oculist to the Children's Hospital, Melbourne.

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American Ophthalmological Society

THE following officers were elected at the last annual meeting of this Society: President: Dr. Hiram Woods, Baltimore; Vice-President: Dr. John E. Weeks, New York; Secretary-Treasurer: Dr. T. B. Holloway, Philadelphia.

* * *

An Interesting Relic

IN welcoming the incoming President of the American Ophthalmological Society at the last meeting of the Society, the outgoing President (Dr. Lucien Howe), presented a gavel with a suitably inscribed cylinder made from an old chair or stool that had stood for many years in the office of the late Dr. Noyes, in New York, where the first meeting of the Society was held.